

**OBESITY-RELATED CANCERS AND THEIR RELATIONSHIP TO  
PHYSICAL ACTIVITY AND DIETARY INTAKES**

A Thesis

Presented in Partial Fulfillment of the Requirements for  
Graduation with Distinction in the School of Allied  
Medical Professions of The Ohio State University

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## ABSTRACT

### **Background:**

Current evidence has shown a link between increasing rates of cancer and obesity.

Certain lifestyle habits, including physical activity and dietary intakes, may be potential risk factors for developing certain types of cancers.

### **Methods:**

Data were collected from the 1999-2006 National Health and Nutrition Examination Survey (NHANES). NHANES is a survey research program conducted by the National Center for Health Statistics which incorporates interviews and physical examinations to collect data from this nationally representative population. In this study, adults >45 years were analyzed to assess the differences in lifestyle behaviors among those with a history of an obesity-related cancer (OBRC, n=821), a non-obesity-related cancer (NOBRC, n=637), or no cancer history (NC, n=8860). Physical activity was tabulated for the frequency (activities per week) and duration (minutes per week) of moderate, vigorous and total physical activity. Dietary intake data were collected using a 24-hr recall method; total nutrients and MyPyramid estimates were compared to recommended intake levels.

### **Results:**

There were significantly lower intakes of carbohydrates, niacin, vitamin B6, vitamin E, phosphorus, copper and selenium in the OBRC versus the other two groups ( $P<0.05$ ).

Those with an OBRC were significantly less likely to meet the recommended intakes of calcium and vitamin B12 ( $P < 0.05$ ). No significant differences existed in the frequency and duration of physical activity across the three cancer groups.

**Conclusions:**

Several dietary habits should be addressed through cancer prevention programs to minimize the development of obesity-related cancers.

## DEDICATION

Dedicated to my family.

Thank you for all of your love and encouragement.

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I would first like to extend my utmost gratitude to Dr. Christopher Taylor. It was through his dedication in helping me investigate my questions and teaching me how to carry out challenging research that I was able to complete this project. Dr. Taylor was patient with me throughout this entire learning process. Without the strong letters of recommendation, I would not have been able to receive the scholarship and grant that I was awarded for doing this distinction thesis. The guidance he provided and the vast amount of time he set aside for this project truly means a lot.

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## INTRODUCTION

### Background

Cancer is the second leading cause of death in the U.S. The American Cancer Society statistics reveal an increased prevalence in cancer across the United States. It is estimated that in 2009 there will be 1.47 million new cancer diagnoses, which has increased considerably from 1.27 million in 2001. The number of cancer deaths for 2009 is estimated at 562,340, which has increased from 559,650 in 2007 (1, 2).

The rates of obesity in the U.S. have consistently risen over the past few decades, as there has been an estimated 9.5% increase since 1988. Overweight and obesity are defined as an individual having a body mass index (BMI) of greater than 25 and 30 kg/m<sup>2</sup>, respectively. Data from 1988-1994 showed that 22.9% of adults over 20 years of age were obese. Then in the 2003-2004 data, this number of obese adults increased to 32.2%. This increase represents one-third of U.S. adults who are considered clinically obese.

Current evidence suggests a significant relationship between the increase of obesity and the rise in cancer rates (4,5). The cancers with the top five mortality rates include lung, breast, colon, prostate, and pancreatic cancer. Of these, obesity has been found to play a role in all but lung cancer (4). Epidemiological studies have established an association between obesity and adenocarcinoma of the esophagus, colon, kidney, and post menopausal breast cancer. Research has also suggested the link between obesity and increased risk for Hodgkin lymphoma, pancreatic, thyroid, ovarian, cervical, prostate, gallbladder, and thyroid cancers (5).

Lifestyle factors such as dietary habits and physical activity have been linked to the development of obesity as well as an increased risk of developing cancer. It is estimated that approximately one-third of the 500,000 cancer deaths that occur each year are related to

lifestyles factors such as physical activity levels and dietary intakes (5). Nutrition and Physical Activity Guidelines, which were established by the American Cancer Society, emphasized weight control, physical activity and dietary habits to reduce the risk of cancer (5). Thus, a further examination of the lifestyle behaviors related to cancer risk by cancer history would provide important formative data for targeted cancer prevention efforts.

### **Problem Statement**

As the number of obesity-related cancers is continually increasing each year in the U.S., it is evident that understanding the underlying risk factors such as physical activity levels and dietary habits will not only aid cancer prevention efforts but could also potentially decrease the mortality rates from cancer (1). In the dietetics field, it is important to assess these risk factors to provide proper medical nutrition therapy (MNT) and aid individuals to decrease the obesity rates and prevent cancer.

It is estimated that overweight and obesity contribute to 14% to 20% of cancer mortalities (5). There are several biological mechanisms which may contribute to either the development of cancer or cancer mortality. Effects of sugar and fat metabolism, compromised immune function, hormone levels (i.e. insulin and estradiol), cell proliferation and growth factors (i.e. insulin-like growth factor-1) are a few of the many physiological changes caused by overweight and obesity.

### **Objectives**

1. To explore the relationship among moderate, vigorous, and total physical activity and history of obesity-related cancers.
2. To explore the relationship between history of obesity-related cancer and dietary intake habits.

## **REVIEW OF LITERATURE**

There have been a number of studies which have examined physical activity levels and the relationship to developing cancer. It has been suggested that exercising 60 minutes 5 or more days a week may have an indirect effect on developing certain obesity-related cancers (5). A case-control study of pre and post menopausal women found that those women who exercised an average of 4 hours per week for at least 12 years, women who exercised at a high intensity for the last 10 years, and those who exercised throughout their lifetime were at a significantly lower risk of developing breast cancer than those who did not exercise. However, the protective effect of exercise was minimized in women who gained a significant amount of weight during adulthood (6). Although healthy activity patterns are often developed during childhood, increasing physical activity at any age in the life cycle can reduce risks of developing cancer (5).

Evidence suggests that the most consistent risk factor linked to colon cancer has been physical activity (7). An examination of occupational and leisure time physical activity reported a 50 percent decreased risk of developing colon cancer in those who had the highest activity levels (8). In two U.S. prospective cohort studies, the Health Professionals Follow-up Study and the Nurses Health Study, results have shown a significant relationship among obesity, physical activity, and the development of pancreatic cancer. An inverse relationship between physical activity and pancreatic cancer was found among those who engaged in moderate activity. For individuals with a BMI less than 25, total physical activity was not associated with an increased risk; however, those with a BMI greater than 25 showed a higher risk for developing pancreatic cancer (9).

Several dietary habits have been shown to modify the risk for developing certain types of cancers. Certain nutrients such as fiber and fat have demonstrated a relationship to the risk of

certain cancers (10,11). For example, fiber has been strongly linked to the decreased risk of colon cancer due to reduced colon transit time, the effects of fiber on absorption of gastrointestinal carcinogens, and altering bile acid metabolism. Women from the Nurses Health Study were assessed for their dietary intakes and results showed that those who had the greatest consumption of fruit fiber had a decreased risk for the development of colon cancer (10). Higher fat intakes may increase the risk for breast cancer in post-menopausal women. The mechanism of fat has been shown to increase blood estrogen levels which are evident in breast cancer. A secondary mechanism of fat is that it may indirectly contribute to obesity (i.e. fat is calorically dense) and eventually lead to cancer (11).

Consuming a diet that consists of fruits and vegetables may affect the risk of developing obesity-related cancers for reasons that relate to obesity prevention because these foods provide many nutrients including fiber and antioxidants. These foods provide anti-oxidants, such as vitamin C, and other chemical properties that are protective against certain cancers (7). Greater consumption of fruits and vegetables has been correlated with increased physical activity and less meat consumption. Also, having a diet high in fruits and vegetables has been associated with a decreased risk of lung, esophageal, stomach, and colorectal cancers (5).

## **MATERIALS AND METHODS**

### **Sample Data**

Data from the 1999-2006 National Health and Nutrition Examination Survey (NHANES) were used to assess the relationships between modifiable lifestyle behaviors and history of obesity-related cancers. NHANES examines about 5,000 individuals a year to collect information on their health and nutritional status. The examinations of these individuals are carried out by trained health professionals who conduct interviews and physical examinations. Physical examinations consist of vision, hearing, medical and dental examinations, physical measurements (i.e. body composition) and laboratory tests. The interviews include demographic, socioeconomic, dietary, and health-related questions.

This complex, multi-stage sample is representative of those adults and children in the U.S. Oversampling of specific hard-to-reach populations, including low-income, children, adolescents, elderly, pregnant women, Mexican Americans and African Americans, ensure adequate representation of commonly underrepresented groups. The significant strength of NHANES is demonstrated in their assessment of chronic conditions that have been diagnosed and undiagnosed as well as looking at risk factors (12).

### **Subjects**

We examined data from adults over the age of 45 years in the 1999-2006 NHANES samples. The women were excluded if they were pregnant or lactating. Information was also provided on current and past medical conditions, BMI, physical activity and dietary recall data. To account for diet and physical activity changes that may result from cancer treatment, those individuals who had been diagnosed with cancer in the last year were excluded from this study.

## **Purpose of the Study**

Because of the recent increases in both cancer and obesity rates as well as the documented impact of lifestyle behaviors on each, the purpose of this study was to demonstrate how low physical activity levels can lead to an increased risk of developing obesity-related cancers. Current evidence suggests that physical activity is inversely related to cancer risk; however, upon investigating the specific intensities and duration of physical activity, there emerged an enhanced understanding of how activity affected those risks.

Another goal of this study was to show how energy intake, nutrient intake, and physical activity all relate to an increased risk of developing obesity-related cancers. Recent research demonstrated how weight loss had shown an improvement in some of the cancer mechanisms such as cell growth, cell proliferation, altered hormone metabolism, and immune system function. Therefore, those individuals who were obese were at an increased risk for these obesity-related cancers because of their decreased immune function and altered cell and hormone metabolism (5).

## **Data Preparation**

Public use data files were downloaded from the NHANES website and imported into SPSS (version 17.0, SPSS, Chicago, IL) for further analysis. Some transformation of existing data was necessary to evaluate the subjects' lifestyle behaviors by cancer history.

**Cancer history** data collected was based on self-reported medical conditions questionnaires obtained during the visit to the Mobile Examination Center (MEC). The subjects were asked if they have ever been told by a health care professional whether they had any chronic diseases, including cancer. If cancer history was reported, data was further evaluated for the type of diagnosed cancer to see if it met the criteria for this research study. The information



collected was recoded into three groups: those with self-reports of no cancer history (NC), non-obesity related cancer history (NOBRC) and obesity-related cancer history (OBRC). For cancer history subjects, the self-reported cancers were compared to the table of obesity-related cancers listed below:

Bone	Hodgkin Lymphoma
Breast (post menopausal)	Ovary (ovarian)
Cervix (cervical)	Pancreas (pancreatic)
Colon	Prostate
Esophagus (esophageal)	Rectum (rectal)
Gallbladder	Thyroid
Kidney	Uterus (uterine)

**Table 1. List of Obesity-related Cancers**

Age of cancer diagnosis was compared to current age to estimate length, since diagnosis was at least one year. For women reporting a history of breast cancer, age at diagnosis was compared to the self-reported age on menopause to identify post menopausal breast cancer.

**Physical activity** was based on self-reported data from questionnaires collected at the MEC. Subjects were asked about the activities they performed over the past 30 days. For each activity reported, the intensity (vigorous or moderate), frequency, and the usual duration of activity were collected. Vigorous activities were defined as those that cause heavy sweating or a large increasing in breathing or heart rate whereas moderate activities were those activities that caused light sweating or a slight to moderate increase in breathing or heart rate. For activities reported, the frequency (times/week) and duration (minutes/week) of moderate, vigorous, and total activities were computed.

**Dietary intakes** were collected using 24-hour recalls administered by trained interviewers. The U.S. Department of Agriculture (USDA) provided estimated nutrient and

MyPyramid intakes for participants. Data collected from the 24-hour recall was assessed based on total intake of calories (kcal), macronutrients, micronutrients and then compared to MyPyramid estimates. To improve comparisons across those with varying amounts of food intakes, nutrient density were computed for nutrients using the following formula:

$$\text{total nutrient consumed} / \text{total energy consumed} \times 1,000 = \text{nutrient} / 1,000 \text{ kcal}$$

For example, intakes of calcium were expressed as calcium (mg) per 1,000 kcal consumed.

### **Data Analysis**

We analyzed this nationally representative sample to identify differences in physical activity and dietary intakes by cancer history. Analysis of variance (ANOVA) was computed to compare the frequency and duration of physical activity and dietary intakes across cancer diagnosis groups. Mean differences in the frequencies and durations of moderate, vigorous, and total physical activity were tested among those individuals with no cancer history, a non-obesity related cancer, and an obesity-related cancer (Objective 1). Differences in dietary intakes were analyzed using total energy intake, energy-adjusted macronutrients (nutrient/1000 kcal), micronutrients (nutrient/1,000 kcal), and total MyPyramid intakes from the meat, milk, fruit, vegetable, grain and whole grain groups (Objective 2).

This data was tabulated for analysis using SPSS (version 17.0). All analyses were conducted using the SPSS Complex Samples (version 17.0) to account for the stratified, multi-staged sampling technique used in subject selection.

## **RESULTS**

### **Total Nutrient Intakes**

Total nutrient intakes were compared to identify differences by cancer history groups (Table 2). Those with an OBRC had the lowest energy intakes ( $P<0.001$ ). Those in the OBRC group had significantly lower intakes of the following macronutrients when compared to the NOBRC group and the NC group: calories ( $P<0.001$ ), protein ( $P<0.001$ ), carbohydrates ( $P<0.001$ ), fiber ( $P=0.007$ ), and fat ( $P<0.001$ ). A lower total intake of micronutrients also existed in the OBRC group. Total riboflavin ( $P=0.026$ ), niacin ( $P<0.001$ ), vitamin B6 ( $P=0.006$ ), vitamin E ( $P=0.004$ ), phosphorus ( $P<0.001$ ), magnesium ( $P=0.003$ ), zinc ( $P<0.001$ ), copper ( $P=0.001$ ), sodium ( $P<0.001$ ), and potassium ( $P=0.001$ ) were all lower in the OBRC group when compared to the other two cancer groups. Thiamin ( $P=0.042$ ) was the only micronutrient intake that was significantly lower in the OBRC group than the NOBRC group. Vitamin A ( $P=0.002$ ) was significantly higher and selenium ( $P<0.001$ ) was significantly lower in the OBRC group than the NC group.

### **Proportion of Recommended Intakes**

To assess dietary adequacy, analyses were performed to determine the proportion nutrient intakes compared to recommended intake levels (Table 3). There were several significant differences in the proportion of intakes to recommended levels by cancer history. Those with a history of an OBRC had significantly lower proportions of recommended intakes for carbohydrates ( $P<0.001$ ), niacin ( $P=0.001$ ), vitamin B6 ( $P=0.001$ ), vitamin E ( $P=0.004$ ), phosphorus ( $P<0.001$ ), and copper ( $P=0.001$ ) than those with a NOBRC history. However, those with a history of OBRC also had a significantly higher proportion of recommended intakes for vitamin A ( $P=0.035$ ). Between the OBRC group and the NOBRC groups, there were no

significant differences between proportion of recommended intakes for thiamin ( $P=0.111$ ), riboflavin ( $P=0.052$ ), vitamin C ( $P=0.58$ ), and magnesium ( $P=0.167$ ).

Calcium ( $P=0.028$ ), zinc ( $P=0.049$ ), and selenium ( $P<0.001$ ) in the OBRC group were significantly different from those with NC history. The proportion of recommended intakes were significantly lower for those with an OBRC when compared to those with NC history. The proportion of recommended intakes for thiamin, riboflavin, vitamin C, and magnesium were not significantly different between the OBRC and NC groups.

There were significantly higher proportionate intakes of two nutrients: dietary fiber ( $P=0.007$ ) and iron ( $P=0.015$ ) in the NOBRC group than those with NC history. No significant differences existed for dietary thiamin ( $P=0.111$ ), riboflavin ( $P=0.052$ ), folate ( $P=0.055$ ), vitamin C ( $P=0.58$ ), and magnesium ( $P=0.167$ ) among all three of the cancer groups.

### **Proportion Not Meeting Recommended Intakes**

Significant differences existed in the percent of the OBRC group not meeting the recommendations for several nutrients (Table 4). Those in the OBRC group were less likely to meet the needs of vitamin B6 ( $P=0.017$ ), vitamin B12 ( $P=0.013$ ), vitamin E ( $P=0.003$ ), calcium ( $P=0.027$ ), and copper ( $P=0.005$ ). Conversely, the OBRC group had a high proportion of those meeting the recommendations for riboflavin ( $P<0.001$ ) when compared to the NOBRC or NC groups.

### **Physical Activity**

No significant differences in existed among all three cancer groups in the frequency (x/week) or duration (min/week) of physical activity (Table 5). The frequency of physical activity in the moderate ( $P=0.121$ ), vigorous ( $P=0.427$ ), and total activity ( $P=0.663$ ) were not

significantly different. Also, the duration of activity in moderate ( $P=0.989$ ), vigorous ( $P=0.086$ ), and total ( $P=0.307$ ) activity did not differ between the cancer groups.

	Total		OBRC		NOBRC		NC		P
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	
Energy (kcal)	1988	13	1747	29	1986	45	2008	15	<.001
Protein (gm)	76.4	.6	66.9	1.2	74.1	1.5	77.4	.7	<.001
Carbohydrate (gm)	240	2	218	4	243	6	241	2	<.001
Total fat (gm)	76.6	.6	65.9	1.4	78.2	2.2	77.4	.7	<.001
Cholesterol (mg)	276	3	249	9	264	11	280	3	0.115
Dietary fiber (gm)	15.7	.2	14.9	.4	16.7	.6	15.7	.2	0.007
Vitamin A (RE)	711	11	759	30	710	30	707	12	0.002
Thiamin (mg)	1.6	.02	1.5	.03	1.6	.04	1.6	.02	0.042
Riboflavin (mg)	2.1	.02	2.0	.04	2.2	.05	2.1	.02	0.026
Niacin (mg)	22.5	.2	20.6	.4	22.4	.5	22.6	.2	<.001
Vitamin B6 (mg)	1.8	.02	1.7	.04	1.9	.04	1.8	.02	0.006
Total Folate (mcg)	385	4	372	7	397	10	385	5	0.055
Vitamin B12 (mcg)	5.1	.1	5.2	.4	5.2	.3	5.1	.1	0.059
Vitamin C (mg)	89.8	1.7	86.6	3.2	89.2	4.0	90.1	1.8	0.834
Vitamin E (mg ATE)	7.4	.1	6.9	.2	7.9	.3	7.4	.1	0.004
Calcium (mg)	806	9	751	18	829	24	809	10	0.114
Phosphorus (mg)	1235	9	1116	19	1247	28	1244	10	<.001
Magnesium (mg)	280	2	260	6	289	7	281	3	0.003
Iron (mg)	15.1	.2	14.6	.3	15.6	.4	15.1	.2	0.077
Zinc (mg)	11.5	.2	10.2	.2	11.4	.3	11.6	.2	<.001
Copper (mg)	1.3	.02	1.2	.03	1.3	.04	1.3	.02	0.001
Sodium (mg)	3201	23	2791	50	3194	84	3235	28	<.001
Potassium (mg)	2715	21	2502	46	2795	62	2726	23	0.001
Selenium (mcg)	102.6	.8	92.2	2.3	99.1	2.6	103.7	.9	<.001

**Table 2: Differences in the Total Raw Nutrient Intakes by Cancer History**

<b>Percent of DRI</b>	<b>OBRC (n=776)</b>		<b>NOBRC (n=607)</b>		<b>NC (n=8,471)</b>		<b>P</b>
	<b>Mean</b>	<b>SE</b>	<b>Mean</b>	<b>SE</b>	<b>Mean</b>	<b>SE</b>	
Carbohydrate (gm)	218.5	4.1	242.9	5.6	241.4	2.1	<0.001
<i>Dietary fiber (gm)</i>	62.4	1.7	65.3	2.1	61.4	0.7	0.007
Thiamin (mg)	159.8	3.2	171.3	4.6	164.6	1.8	0.111
Riboflavin (mg)	209.7	4.3	220.0	4.7	211.2	2.1	0.052
Niacin (mg)	181.1	3.2	194.0	4.3	196.0	2.0	0.001
Vitamin B6 (mg)	131.6	2.9	142.9	3.2	144.2	1.7	0.001
Total Folate (mcg)	116.3	2.3	123.9	3.1	120.4	1.5	0.055
Vitamin B12 (mcg)	261.9	21.4	260.8	15.5	256.0	4.8	0.039
Vitamin C (mg)	133.2	5.0	132.6	5.9	135.1	2.7	0.580
Vitamin A (RE)	389.2	33.7	271.6	21.3	367.0	14.7	0.035
Vitamin E (ATE) (mg)	57.2	1.7	65.8	2.5	61.9	0.7	0.004
<i>Calcium (mg)</i>	64.0	1.5	70.5	2.1	71.2	0.9	0.028
Phosphorus (mg)	192.4	3.3	215.0	4.8	214.5	1.6	<0.001
Magnesium (mg)	88.6	2.0	93.5	2.2	92.1	0.8	0.167
Iron (mg)	261.6	5.8	276.3	6.5	260.6	3.0	0.015
Zinc (mg)	132.4	3.0	140.1	3.7	144.1	2.1	0.049
Copper (mg)	167.6	4.2	183.6	5.0	184.5	2.5	0.001
Selenium (mcg)	204.8	5.1	220.3	5.9	230.5	1.9	<0.001

Nutrients in regular type represent Estimated Average Requirements (EAR)

Nutrients in *italics* are Adequate Intakes (AI)

**Table 3: Differences in the Mean Percent of Recommended Intakes by Cancer History**

<b>Nutrient</b>	<b>OBRC</b>	<b>NOBRC</b>	<b>NC</b>	<b>P</b>
Carbohydrate	6.0	5.8	6.6	NS
Dietary fiber	86.8	85.6	87.6	NS
Thiamin	23.8	19.4	22.3	NS
Riboflavin	7.6	8.0	11.4	<0.001
Niacin	16.4	13.4	14.6	
Vitamin B6	39.7	35.1	34.4	0.017
Total Folate	50.5	42.0	45.7	0.013
Vitamin B12	24.9	21.0	23.6	
Vitamin C	50.4	48.5	52.1	
Vitamin A	39.6	44.3	45.5	
Vitamin E	89.9	84.3	86.2	0.003
Calcium	83.6	81.2	79.5	0.027
Phosphorus	10.1	8.3	9.6	0.005
Magnesium	67.3	64.8	64.1	
Iron	7.1	5.5	7.6	
Zinc	38.2	35.3	35.7	
Copper	21.4	14.3	17.6	
Selenium	13.4	11.0	10.2	

**Table 4. Proportion Not Meeting Recommended Intake Levels by Cancer History**

<b>Physical Activity</b>	<b>OBRC (n=393)</b>		<b>NOBRC (n=346)</b>		<b>NC (n=4389)</b>		
<b>Frequency of activity (times/wk)</b>	<b>Mean</b>	<b>SE</b>	<b>Mean</b>	<b>SE</b>	<b>Mean</b>	<b>SE</b>	<b>P</b>
Moderate activity	5.2	0.4	4.8	0.3	4.4	0.1	0.121
Vigorous activity	1.7	0.3	1.9	0.3	2.1	0.1	0.427
Total activity	6.8	0.5	6.7	0.3	6.5	0.1	0.663
<b>Duration of activity (min/wk)</b>							
Moderate activity	201	14	204	13	203	6	0.989
Vigorous activity	76	11	90	15	105	8	0.086
Total activity	278	19	294	20	308	9	0.307

**Table 5: Differences in the Mean Levels of Frequency and Duration of Physical Activity by Cancer History**



## **DISCUSSION**

Lifestyle factors, including dietary intakes and physical activity habits, play an important role in the development of various types of cancers, both obesity and non-obesity related (5). About one-third of all cancers can be prevented by incorporating proper lifestyles factors into daily life (5, 13). Although there are no randomized controlled trials investigating the specific relationships among physical activity, dietary intakes, and obesity-related cancers, this study may serve as preliminary evidence to support more research in the area relating to cancer prevention efforts.

Of the top 5 most common cancers, four of the obesity-related cancers include breast, colon, prostate, and pancreatic. Foods which include nutrients such as fiber, fat, calcium, copper, and antioxidants have shown to play a role in protection against these cancers (4). Although there were several macronutrient and micronutrients that differed among the three groups in this study, some of the lower intakes may be related to lower total energy intakes. For example, the OBRC group had a lower total intake of fiber (14.9 gm/day); however, their total mean energy intakes were lower (1747 kcals/day) when compared to the other two groups. When nutrient intakes were evaluated, it was important to consider the total energy intake and the proportion of that nutrient to the total energy intake.

Even though there were no significant differences in fiber intake across the three cancer groups, the data still shows that there was an overall lower intake per day than suggested by the established recommended intakes. On average, 86.6% of all the individuals did not meet the recommended intakes for fiber. A body of evidence exists for high fiber intakes being protective against colon, rectal, and esophageal cancers (5, 10). Foods that are high in fiber may also lead to a decreased chance of becoming obese because these foods are typically lower in calories and

move more slowly through the digestive tract. A fiber intake which meets the recommendations may decrease the risk for exceeding energy needs, which may lower obesity and cancer rates (5).

When the OBRC group was compared to the NOBRC and NC groups, those individuals with an OBRC had a significantly lower proportion of total nutrient and recommended intakes for carbohydrates, niacin, vitamin B6, and vitamin B12. Vitamins B6 and B12 are enzymatic cofactors in folate metabolism, which ultimately play a role in DNA methylation, repair, and genetic mutations (14). Although there is discussion that these B-vitamins are not associated with an increased cancer risk, specifically pancreatic cancer (15), the results from this investigation may provide evidence for a possible association of vitamins B6 and B12 with obesity-related cancers.

Cancer Prevention Guidelines established in 2009 by the American Cancer Society suggest the importance of obtaining a healthy, plant-based diet to maintain a healthy weight and protect against disease (16). Through a high fruit and vegetable diet, individuals can maintain an appropriate antioxidant intake as well as fiber. These two groups of nutrients are important in maintaining a healthy weight and preventing disease (5). It remains controversial as to whether vitamin E, an antioxidant, is protective against prostate cancer, although the significant findings in this study may provide otherwise because the OBRC group had 89.9% of individuals who were not meeting the recommended intakes (versus NOBRC = 84.3%, NC = 86.2%). Selenium, another antioxidant, which was significantly lower in the OBRC group, is still being debated in its role in cancer development. These two antioxidants may play a crucial role in cancer development; however, further research is needed to confirm this relationship of these specific nutrients to cancer.

Although there were significantly lower intakes of copper and phosphorus in the OBRC group, current research has not suggested that these nutrients play a major role in the development of cancer. More investigations relating to these specific nutrients should be done to further identify their relationships to cancer.

Calcium intake was significantly lower in the OBRC group with 83.6% of individuals not meeting the recommended intake (versus NOBRC = 81.2%, NC = 79.5%). Studies have shown that a high calcium intake is associated with a decreased risk for all cancers in healthy, post menopausal women (17). However, some studies have linked high calcium intakes to an increased risk for prostate cancer while others have demonstrated the opposite correlation (18, 19). This remains to be controversial in relation to cancer development and whether higher or lower intakes exhibit protective effects.

Current Physical Activity Guidelines, which have been determined by the U.S. Department of Health and Human Services, recommends that adults maintain 150 minutes per week of moderate-intensity aerobic activity or 75 minutes of vigorous-intensity activity in addition to normal daily activities. For more extensive health benefits, these guidelines suggest that adults increase their moderate-intensity aerobic activity to 300 minutes per week and vigorous-intensity aerobic activity to 150 minutes per week (20).

Even though there were no significant differences in physical activity among the three cancer groups, the data shows that all of the groups were exceeding the established physical activity guidelines for duration of moderate and vigorous activities. Total duration of activity ranged from 278 total minutes to 308 total minutes on average per week among all three groups. The evidence also demonstrates that all three groups met the minimum recommendations for moderate and vigorous activities. However, none of the groups are reported to be meeting the

recommendations for more extensive health benefits. Interestingly, the NC group had the highest amount of total duration each week (308 min/wk) and the highest amount of vigorous activity (105 min/wk). These findings may indicate that the increased amount of vigorous activity and total activity was protective against cancer.

Several limitations existed in this study. Study participants in the NHANES surveys were administered 24-hour dietary recalls, which could be limited by under reporting or over reporting of nutrient intakes because of reliance on memory. Nutrient intakes may not reflect historical dietary intakes that may have contributed to cancer development because of the design of the 24-hour dietary recalls. However, the data provided in this study may indicate the participant's current dietary habits which may not be conclusive in investigating the relationship between diet and cancer development. With this study design, it was difficult to disseminate which lifestyle factors are related to the onset of obesity, the development of cancer, or both.

There could have been errors in the physical activity reporting by participants also. Recall bias could have skewed the frequency and duration data of physical activities. Even though there were no differences among the three groups, evidence shows that increased physical activity contributes to a decreased risk of cancer and obesity (5, 21).

## **CONCLUSIONS AND APPLICATIONS**

Additional research is required to further identify specific lifestyle factors that contribute to obesity-related cancers. Although this is a complex disease and so many variables exist which include modifiable and non-modifiable risk factors, the data analyzed in this study provides more evidence for future research.

Extensive evidence on the relationship of obesity to cancer and the roles that lifestyle factors play may provide a basis for the development of cancer prevention programs across the U.S. If possible, prospective randomized controlled trials including long-term intervention studies are needed to establish individualized guidelines for all populations. Studies which identify specific dietary and physical activity recommendations are needed to assist in efforts to prevent both obesity and cancer. It would also be beneficial to further evaluate when it would be most effective in the life cycle to incorporate these recommendations to provide the most extensive protection against certain cancers. This growing body of evidence may benefit those individuals with a pre-disposition to cancer by aiding in preventative efforts. It can also benefit those individuals with pre-existing cancer by providing recommendations that would help to decrease the mortality rates.

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